BOXER'S HEMORRHAGE*

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DISCUSSION *by Walter F. Schaller, M.D., San Francisco; Edmund J. Morrissey, M.D., San Francisco.*

MULTIPLE petechial hemorrhages associated with trauma of the brain have frequently been observed and reported, both in experimental animals and in man during the past five decades. Jakob,[1] in repeating Schmaus'[2] work, produced petechial hemorrhage in the brain and cord of rabbits and monkeys by subjecting the animals to minor blows which were designed to produce only concussion. He did not believe that these capillary hemorrhages were the result of contusion directly, but that they were only secondary to glial degeneration following concussion and its sequences. That petechial hemorrhage in the brain will follow concussion alone, without contusion of the brain substance, has also been reported by Ricker[3] and Cassasa.[4] The latter encountered five instances of multiple traumatic cerebral hemorrhage without associated fracture of the skull, and he attributed the bleeding to sudden overfilling of the perivascular spaces with cerebrospinal fluid, which, in turn, produced laceration of the vessels by tearing their walls in the neighborhood of the fibrillary attachments to the surrounding brain tissue. Osnato and Giliberti[5] reported a case of fatal injury of the brain in which the period of survival was thirty-six hours, and there was no fracture of the skull. They found petechial hemorrhages scattered through the centrum ovale, the corpus callosum and pons. Martland and Beling,[6] following Cassasa's theory, reported twenty-three instances of multiple deep hemorrhages in 309 cases in man, in which the outcome was fatal. In nine of these cases petechial hemorrhages were found. Winkelman and Eckel[7] reported seven cases of severe injury of the head in which there was microscopic study of the brain, and announced petechial hemorrhages in five of these. These authors did not commit themselves definitely as to the nature of the traumatic petechiae. Rand and Courville[8] studied the brain fibers and the glial reaction in relationship to petechiae. Endbulbs, corkscrew twisting and fragmentation of nerve fibers surrounded these hemorrhages. In the smaller hemorrhages there was no noticeable change in the glia, but in the larger hemorrhages regressive changes were produced by compression, local ischemia and softening, leaving a small, cyst-like space surrounded by more or less active proliferating glia. Glial rings were thus produced. Schaller[9] and his coworkers published a series of examinations on the human brain and experiments on the albino rat, through which they found deep petechial hemorrhages present commonly, but did not believe that the mechanism of the ordinary forms was satisfactorily explained by traumatic tearing of the vessel. They concluded that deep traumatic petechiae were dependent chiefly on the effects of vasomotor concussion, causing vasodilatation, prestasis, anoxemia, impairment of the walls of the vessels, diapedesis and perivascular hemorrhage. Petechial hemorrhages are also known to follow not only indirect mechanisms of the sorts described above, but to occur as a sequence to contusion or bruising; this term being preserved for lesions produced at the instant of trauma and not confused with concussion in which the sequences require some time to develop. Gonzales, Vance and Helper[10] have found multiple small hemorrhages of petechial size associated with violence producing a sudden jarring of the head, but still not of sufficient moment to cause a fracture of the skull. They indicate that in these cases the brain is made to oscillate, and contuses itself against the skull and the different reflections of the dura like the falx cerebri and tentorium. For example, they have found multiple hemorrhages produced in the brain stem after an impact in the region of the parietal boss. They believe that such a movement causes the pons and cerebral peduncles to be injured against the sharp edges of the tentorium, sometimes to the point of complete laceration. On the other hand, the impacts against the lateral frontal region were observed to produce hemorrhages in the white matter of the frontal lobes, and in the basal nuclei. They found the corpus callosum could be involved by impacts over the frontal region in anteroposterior direction, causing that structure to be contused or completely severed by the sharp edge of the falx cerebri. In some instances they believe the oscillation of the brain inside the skull might give rise to punctate hemorrhages throughout both the grey and the white matter of the cerebral hemisphere. In 1931, Moody[11] reported a series of contusion hemorrhages of the brain in which, out of seventy-eight brains examined and showing varying degrees of contusion and hemorrhage, only forty-two were taken from dead persons who had fracture of the skull, and several had no evidence of injury to the scalp or pericranial tissue. In collecting his series at that time, one case was encountered occurring in a boxer where, following a severe beating, the individual died and at autopsy showed scattered, small, contusion-like hemorrhages in the cortex, cerebellum and stem. In the ensuing eight years we have collected a series of three more cases of contusion hemorrhages of the brain occurring in the stem in which there was no fracture of the skull, and no injury elsewhere in the brain, either cerebral or cerebellar.

REPORT OF CASES

CASE 1.—The patient was engaged in a professional boxing match in Oakland, California, the evening of August 26, 1930. At 10:15 o'clock, during the second round of the encounter, he was struck, apparently on the jaw, and knocked to the canvas. He arose after a short interval and was able to continue and finish the round. During the following two rounds he seemed somewhat dazed and to have lost some coordination, but he managed to survive the two rounds successfully. All this time, however, his head was struck re-

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peatedly by his opponent, rocking it back and forth and from side to side. In the fifth round he was again struck a sharp blow, which apparently caused a momentary loss of consciousness, and he turned clinging to the rope. At this point he was struck again sharply from behind just below the occiput, forcing his chin down suddenly and abruptly upon his chest. He slumped upon the ropes and hung there, receiving several more blows before the referee disengaged his opponent. He was carried from the ring, but failed to regain consciousness in his dressing room, so was taken to the hospital that night. The patient remained unconscious, moving occasionally during the night and the next morning, and, while preparations were being made to examine him, he died at 11:35 a. m.

AUTOPSY REPORT:

The necropsy showed a normal young, white male in an excellent state of development. Because of the normal character of the body, excepting the central nervous system, the general autopsy protocol is omitted here. In the central nervous system, however, there was considerable edema, both of the leptomeninges and of the brain. There were scattered small, contusion-like hemorrhages here and there beneath the cortex of the frontal, occipital and parietal lobes, and rather marked hemorrhages of a similar type in the brain stem extending from the base into the medulla. There was no evidence of disease in the blood vessels. The cerebellum also contained scattered small hemorrhages. Histological examination corroborated the gross appearance, and a diagnosis was made of subpial intracerebral and pontine hemorrhages with the characteristics of those which occur as the result of indirect violence.

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CASE 2.—On June 28, 1933, the patient was engaged in a professional fight in Reno, Nevada. During the third round of the fight he was knocked unconscious for ten or fifteen seconds by a sharp blow on the head. After being counted out, he got up and walked to his corner of the ring, seeming at this time to be normal. Two days later, on July 1, while sitting about in the evening, and after a few premonitory groans, he suddenly lost consciousness. He was taken to the hospital immediately, in a deep stupor; and upon entry, there was no response to painful stimuli. The reflexes were about equal on both sides, possibly a little more active on the left. There was a bilateral Babinski, a bilateral ankle clonus which was better sustained on the left than on the right, and both pupils were dilated and fixed. There was a slight muscular resistance in the left leg. All superficial reflexes were absent. The Kernig was suggestive on both sides, but there was no neck stiffness. The patient had alternating periods of restlessness and deep stupor, with respirations irregular and varying, with the periods of restlessness from 30 down to 8 per minute. The pulse was regular, but varied in rate with the respiration from 20 up to 48. A spinal tap was done, with release of frankly bloody fluid under a pressure of 198 millimeters of water. About 15 cubic centimeters were removed, following which the pressure dropped to 100 millimeters. The Queckenstedt was normal. Physical examination was otherwise normal. The patient was given artificial and pump respiration to relieve the respiratory stridor, oxygen inhalation, 100 cubic centimeters of 50 per cent glucose intravenously, and finally placed in a Drinker respirator in an effort to stabilize the respiration and pulse rate. On July 2, four days after injury, a right frontal craniotomy was done without anesthesia. At this time the patient's pulse could not be obtained, and his respiration was carried on solely by the Drinker respirator. When the dura was opened, no pulsations could be seen and the brain did not tend to herniate through the dural opening. There was some blood immediately beneath the dura which seemed to be just coagulating. At this stage in the operation it was evident that the patient was dead, and no attempt was made to clean out what appeared to be hematoma. The incision was closed with a single row of dermal sutures.

AUTOPSY REPORT:

The subject was a young Filipino male in an excellent state of nutrition and development. The body was normal, excepting for the head and central nervous system, and for this reason the general protocol is omitted.

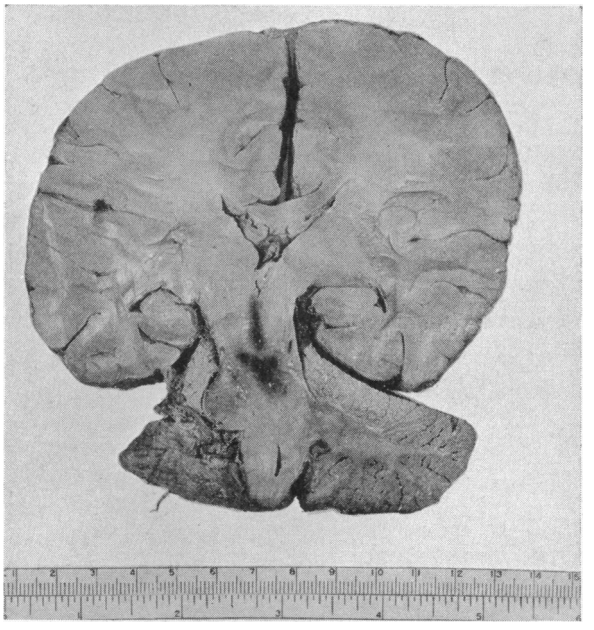

Fig. 1.—Central hemorrhage in the pons.

There was a small, shaved area in the right frontal region, where a horizontal incision had been made. There was a burr hole directly beneath this incision, through which the brain could be seen. The dura had been incised. There was a little blood in the opening. Upon lifting off the cranial cap, a very edematous brain was seen, which was stained with blood about the burr hole, apparently from the surgery. No subdural hematoma was seen. The brain was symmetrical, the convolutions were flat, and the sulci were shallow. The vascular arrangement was normal at the base, and sections through the cerebrum showed normal, symmetrical, undilated ventricles lined with a smooth ependyma. Upon making a longitudinal cut through the stem, fresh hemorrhage was seen arising from several small areas of contusions in the pons and dissecting through the adjacent structure into the medulla (Fig. 1). Blood had passed from these contusions into the fourth ventricle and down the spinal canal.

Microscopic sections showed a definite edema of both the brain and leptomeninges. The circulatory channels were congested. No areas of hemorrhage or degeneration were found in the cerebrum or cerebellum, but in the pons there was an area of fresh bleeding about which was contusion. Adjacent to this larger area of hemorrhage were many small spots of petechial bleeding.

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CASE 3.—The patient was a middle-aged, white woman who for some time had been living under an assumed name with a man, as husband and wife. The two had been drinking, and on the evening before the patient came into the hospital there was some altercation between them, wherein the woman was rather severely beaten about the head. The patient became unconscious shortly after receiving the blows and remained upon the bed until the following afternoon, when she was brought to the San Francisco Hospital in a still unconscious condition. Her breathing at this time was stertorous, rapid and regular, and the pulse was very thready and rapid, though regular. There were several bruises over both eyelids, which were swollen shut, and there was a depressed area over the left zygoma. No abrasions were found on the scalp. On entering the hospital the patient's temperature was 104.5° F., the pulse was 128, and the respiration 52. The blood pressure at this time was 100/70. During the first hour on the ward the blood pressure fell rapidly, and at 4:30 p. m., one hour after entry, it was 80/65.

PHYSICAL EXAMINATION:

No lacerations were found on the scalp. Both eyelids were swollen shut and were ecchymotic. The pupils were

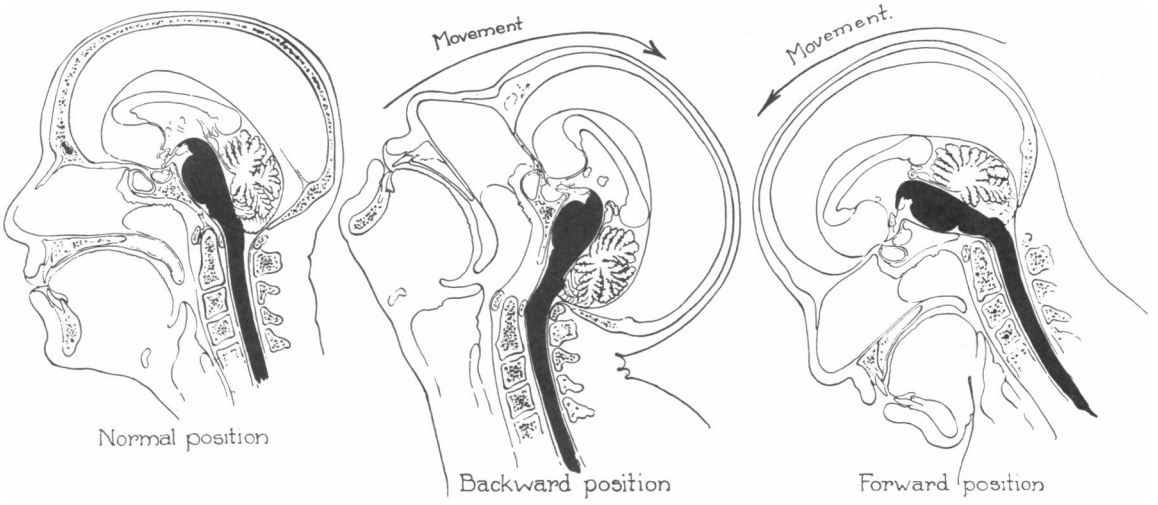

Fig. 2.—Schematic diagram of head movements showingaccentuated flexion of the brain stem.

fixed in contraction and there was no response to light. The conjunctivae were clear. The ears and nose were normal. There was dried blood on the lips and the saliva was also stained a dark red. There was a depression over the left zygoma in the outer half, with swelling of the tissue about that region. The neck was not remarkable. The thorax showed a fair development, was symmetrical, and well formed. There were no areas of dullness in the lungs to percussion, and no râles in the lungs. There were, however, numerous rattling tracheal sounds. The heart was not enlarged, the pulse was thready but rapid, and the heart sounds were muffled by the rattling in the trachea. The tones were of poor quality and the apex beat was in the fifth interspace. The abdomen was soft and flaccid, and striae gravidarum were present. There was an old scar in the midline in the infraumbilical region, but there were no recent abrasions or evidences of injury to the abdomen or the vertebral column. The extremeties were flaccid, as was all of the musculature of the body, and no reflexes could be obtained. A lumbar puncture showed a fluid that was slightly turbid and a little bloody. The pressure was 130 millimeters of water, 140 millimeters at the left jugular compression, 170 millimeters at the right jugular compression, and 190 millimeters upon compressing both veins. The Pandy was 3 plus. There were no white cells in the count and only a few red blood cells were found. Examination of the catherized urine specimen showed a 2-plus sugar, 2-plus acetone, a few fine and coarsely granular casts, no albumin, 6 to 10 pus cells/HDF and a specific gravity of 1,025. The peripheral blood count was normal. Two hours after entry, at 5:55 p. m., the patient died.

AUTOPSY REPORT:

The subject was a white, middle-aged female in a good state of nutrition. External examination showed the physical markings of contusion and depression noted in the history. Excepting for congestion of the lungs and a moderate fatty infiltration of the liver, the viscera were all normal, and the general protocol is omitted here.

Examination of the central nervous system, however, revealed a brain which weighed 1,400 grams and which measured 18x17x10 centimeters. There was a diffuse edema, especially at the vertex, with flattening of the convolutions. The hemispheres were symmetrical and there was a moderate edema of the leptomeninges. No areas of contusion, laceration or hemorrhage were seen on the surface of the brain, and section through the brain showed normal, symmetrical, undilated ventricles lined with smooth ependymae, excepting in the fourth ventricle where the ependymal lining was distinctly granular. Section through the brain stem showed a single area of fresh hemorrhage, 7 millimeters in diameter, in the pons, 3 millimeters above the middle of the aqueduct of Sylvius. Microscopic sections of the brain showed a general edema with some vacuolization and zones of shrinkage around the pyramidal cells. In the stem, tiny fine points of contusion were seen where there had been fresh hemorrhage. There were no evidences of old degeneration or injury.

SUMMARY

These three reports introduce hemorrhages in the brain stem which followed severe beating of the subject, with the individual at least in the terminal stages of the beating only partially conscious or, in the boxing sense, "out on his feet." At such a time the musculature is more relaxed than normal, and muscle tone is definitely decreased. The motion of the head upon the cervical vertebrae is more pronounced than during the usual state, and more acute angulation of the brain stem is possible upon flexion and extension of the head (Fig. 2). Because these first three cases which we encountered have occurred in connection with fisticuffs of some nature, the specific and limited injury is regarded as peculiar to boxers or fighters—to those people who are involuntarily beaten, or to others where the type of injury causes acute angulation, and pinching of the pons and medulla over the tentorium. The term "Boxer's Hemorrhage," therefore, is suggested for these injuries.

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DISCUSSION

WALTER F. SCHALLER, M. D. (909 Hyde Street, San Francisco).—Brain-stem hemorrhages, especially pontile hemorrhages following trauma, have been difficult to explain because of the excellent protection of this region of the brain, lying deep-seated and resting on the water cushion of the basal cisterna. These hemorrhages have been regarded as venous in origin, due to stasis and subsequent rupture of an impaired vessel wall. The stasis has been explained by thrombosis of larger vessels, as the veins of Galen or the venæ striæ terminalis, to which the ruptured one is contributory, or by the mechanism of local vasodilatation and stasis. These explanations receive support from the fact that hemorrhages quite similar to those following trauma occur in inflammatory conditions, and also in secondary to nontraumatic vascular lesions above the tentorium.

The mechanistic explanation of the authors is seductive, and an effort might be made to reproduce these lesions by animal experimentation. It is interesting to note in the article of Kernohan and Woltman on "Incisura of the Crus Due to Contralateral Brain Tumor," *Archives of Neurology and Psychiatry,* February, 1929, that the grooves caused by the pressure of the tentorium are often associated with petechial hemorrhages, as described by the authors. I have maintained (*Archives of Neurology and Psychiatry,* June, July, 1933) that there is no true shifting of posterior fossa contents in the mechanism of hernia into the foramen magnum, for example, as following lumbar puncture. However, the "pinching" described by the authors may be quite a different mechanism. Case 2 of the authors, because of the considerable interval of apparent normalcy after trauma (two days), is somewhat difficult to reconcile with a hemorrhage from acute angulation.

Before full acceptance of this theory several objections must be satisfactorily explained, namely, interval hemorrhage, the lack of occurrence or constancy in extreme cervical displacement—as in diving accidents—and the absence of any other demonstrable evidence of pinching, such as necrosis, laceration, or deformity.

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EDMUND J. MORRISSEY, M. D. (909 Hyde Street, San Francisco).—The autopsy findings reported by Doctors Carr and Moody, of petechial hemorrhages localized especially to the brain stem, are extremely interesting and valuable. They offer an excellent explanation for the variety of symptoms and neurologic findings noted in boxers who have survived several severe beatings in the prize ring, often referred to under the unauthorized but popular term, "punch drunk." The two-day, symptom-free interval in Case 2 would indicate delayed hemorrhages.

Although these petechial hemorrhages of the brain stem, as pointed out, are rather characteristic of the cerebral trauma resulting from blows about the head received during fistic encounters, they do occur in other types of head injuries, and, therefore, the advisability of introducing the term "boxer's hemorrhage" might be questioned.

INTRASPINAL PROTRUSIONS OF THE INTERVERTEBRAL DISCS: ROENTGENOGRAPHIC FINDINGS*

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DISCUSSION *by Carl W. Rand, M. D., Los Angeles; L. H. Garland, M. D., San Francisco; John B. Doyle, M. D., Los Angeles.*

MIXTER and Barr, in 1934, were the first to call attention to the importance of intraspinal protrusions of the intervertebral discs in relation to sciatica and low-back pain. Prior to their report the incidence of protruded discs in the lumbar region was not often recognized, the majority of the reported cases occurring in the cervical region. Their investigation of this condition at the Massachusetts General Hospital, together with the reports by Mixter and Ayer, Hampton and Robinson and Love and Camp, proved conclusively that these lesions were ruptures of portions of the intervertebral discs and the nucleus pulposus into the spinal canal, similar to the posterior protrusions of the nucleus pulposus which Schmorl described and termed "posterior Schmorl's notch."

The clinical and surgical diagnosis of chondromas, enchondromas, ecchondroses, extradural fibrocartilagenous masses, etc., of which Mixter and Ayer in 1935 found forty-seven cases reported in the literature, undoubtedly represent a similar pathologic process.

ROENTGEN DIAGNOSIS

The roentgenographic signs of intraspinal protrusions of the intervertebral discs may be classified as direct and indirect. The direct signs, when present, are of questionable value and do not justify one in making a positive diagnosis. Hampton and Robinson, also Love and Camp, in an analysis of a large series of proved cases of protruded discs, found approximately 40 per cent of their routine spine roentgenograms entirely negative or without any significant changes being present.

NARROWED INTERVERTEBRAL JOINT SPACE

In Camp's series of fifty proved cases of protruded discs, narrowing of the intervertebral space was found in twenty-seven instances; but in nineteen of these the narrowed disc occurred at a different level than the site of the lesion as found at operation.

Both Love and Camp, and Hampton and Robinson, found that the intervertebral spaces between the fourth and fifth lumbar, and the fifth lumbar and upper sacral segment, were the most frequently narrowed, these also being the levels at which the majority of intraspinal protrusions occur. However, when you take into consideration the fact that narrowing of these interspaces occurs with greater frequency than elsewhere in the spine, the conclusion must be that there is no dependable and consistent relationship between a narrowed inter-

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